Magnetic resonance imaging goes postmortem: noninvasive detection and assessment of myocardial infarction by postmortem MRI

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Abstract: OBJECTIVE: To investigate the performance of postmortem magnetic resonance imaging (pmMRI) in identification and characterization of lethal myocardial infarction in a non-invasive manner on human corpses. MATERIALS AND METHODS: Before forensic autopsy, 20 human forensic corpses were examined on a 1.5-T system for the presence of myocardial infarction. Short axis, transversal and longitudinal long axis images (T1-weighted; T2-weighted; PD-weighted) were acquired in situ. In subsequent autopsy, the section technique was adapted to short axis images. Histological investigations were conducted to confirm autopsy and/or radiological diagnoses. RESULTS: Nineteen myocardial lesions were detected and age staged with pmMRI, of which 13 were histologically confirmed (chronic, subacute and acute). Six lesions interpreted as peracute by pmMRI showed no macroscopic or histological finding. Five of the six peracute lesions correlated well to coronary pathology, and one case displayed a severe hypertrophic alteration. CONCLUSION: pmMRI reliably demonstrates chronic, subacute and acute myocardial infarction in situ. In peracute cases pmMRI may display ischemic lesions undetectable at autopsy and routine histology. pmMRI has the potential to substantiate autopsy and to counteract the loss of reliable information on causes of death due to the recent disappearance of the clinical autopsy.

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Abstract *Objective* To investigate the performance of postmortem magnetic resonance imaging (pmMRI) in identification and characterization of lethal myocardial infarction in a non-invasive manner on human corpses. *Materials and Methods* Before forensic autopsy, 20 human forensic corpses were examined on a 1.5-T system for the presence of myocardial infarction. Short axis, transversal and longitudinal long axis images (T1-weighted; T2-weighted; PD-weighted) were acquired in situ. In subsequent autopsy, the section technique was adapted to short axis images. Histological investigations were conducted to confirm autopsy and/or radiological diagnoses. *Results* Nineteen myocardial lesions were detected and age staged with pmMRI, of which 13 were histologically confirmed (chronic, subacute and acute). Six lesions interpreted as peracute by pmMRI showed no macroscopic or histological finding. Five of the six peracute lesions correlated well to coronary pathology, and one case displayed a severe hypertrophic alteration. *Conclusion* pmMRI reliably demonstrates chronic, subacute and acute myocardial infarction in situ. In peracute cases pmMRI may display ischemic lesions undetectable at autopsy and routine histology. pmMRI has the potential to substantiate autopsy and to counteract the loss of reliable information on causes of death due to the recent disappearance of the clinical autopsy.

Keywords Postmortem imaging · Forensic Radiology · Myocardial infarction

Introduction

Over the last decades clinical autopsy rates have been declining dramatically. Being the main source of today's postmortem information, this broadly affects the health of the population and the quality of the health care systems, too. As, for example, national mortality statistics directly influence the financial investments intended to support specific research areas in the health care sector, this development impairs the accuracy of research planning and thereby the expected benefit for the health of the population as well. Efforts to counteract this unfavorable trend have predominantly remained unsuccessful [1–5]. However, possible alternative investigation techniques exist and may have the potential to narrow this information gap.

In the forensic environment, postmortem cross-sectional imaging is being evaluated and validated to assess different forensic findings in a minimally invasive or non-invasive manner mainly for forensic documentation [6–8]. Within these research efforts, it was also experienced that natural causes of death could be visualized in a non-invasive manner. A variety of natural causes of death were exemplarily published by predominantly forensic-radiological research groups [9–16]. It should not be surprising for any physician that macro-morphological findings such as major intra-cerebral bleedings, aortic dissection, pericardial tamponades, tumors, etc., are quite easily diagnosed on

postmortem CT and MRI images. Postmortem imaging is not impaired by patient-related motion artifacts, dose limitations or examination time restrictions. Furthermore, these methods have the potential to provide more detailed information on macroscopic and tissue alterations for the cause of death assessment. In this context, visualization of early ischemic alterations of the myocardium would be most important as cardiac death represents the major portion of the national mortality registers.

The initial cases of myocardial infarction visualized by pmMRI were published from 2003 to 2006 [17–19]. In clinical MR examinations only contrast-enhanced sequences allow sufficient cardiac MR imaging [20]. Postmortem cardiac MR imaging, however, is based on a purely morphological visualization of the investigated structures since only structural tissue alterations influence image contrast. This study focused on cases of myocardial infarction comparing the findings of pmMRI with the pathological findings at autopsy. The aim was to investigate the different stages of myocardial infarction (representing different survival times) in correlation with autopsy and histological findings in a larger study population than that on which the existing literature is based [17–19, 21, 22].

Materials and methods

Cases

Twenty persons dying under circumstances consistent with a cardiac cause of death were prospectively investigated between May 1, 2007 and March 31, 2008. The study population consisted of 16 men and 4 women [mean (±SD) age at death, 63±10 years]. A pmMRI examination of each body was performed before forensic autopsy. The study was approved by the local ethics committee (Dnr M64-05, 75-05).

Logistics

The corpses were undressed and wrapped twice in artifact-free body bags. As the forensic department is not equipped with MR equipment, the imaging infrastructure of a nearby radiological research institute was used based on a pre-existing collaboration. Transportation was carried out using the institutional mortician vehicle. The MR examinations took place between 7 and 8 o'clock a.m. before regular patient scheduling.

MRI

Each corpse was placed supine in a 1.5-T clinical MR machine (Achieva, Philips Medical Systems, Best, The Netherlands) and examined using a SENSE cardiac or a SENSE torso coil. Short axis images were acquired using conventional clinically used localizer settings [23] in T1 (TR 650 ms; TE 15 ms), 2× T2 (TR 4,000 ms; TE 70 ms and TR 4,000 ms; TE 100 ms) and proton density (TR 4,000 ms; TE 10 ms) weighting. Slice thickness was 4 mm, resolution 0.7×0.7 mm. Examination time was <20 min. Image interpretation was performed according to Jackowski et al. [19], predominantly based on the signal behavior on T2-weighted images by two investigators in consensus sessions. Additionally, the finding of hypointensity without marginal hyperintensity in T2-weighted images not yet described by Jackowski et al. [19] was interpreted as peracute infarction.

Autopsy

Forensic autopsy was performed by board-certified forensic pathologists. The cardiac dissection technique was adapted to match short axis images by slicing the ventricles parallel to the heart base. Extensive photographic documentation was carried out. Histological specimens of the entire circumference of the left ventricle (LV) according to the short axis MR images were investigated. Tissue staining included hematoxylin and eosin (H&E), van Gieson and chromotrop-aniline-blue (CAB). The coronary orifices and the apex of the heart were used as anatomical landmarks for comparison of related findings. Histological grading ranged from acute (early and late) to sub-acute (early and late) and chronic.

Results

In 17 of the 20 study cases, cardiac failure was considered the cause of death. In case 12, severe pneumonia was considered to have promoted cardiac failure. Two study cases (9 and 15) presented with an extra-cardiac cause of death.

Sixteen study cases presented with overall 19 myocardial lesions to investigate, and four study cases showed no assessable local ischemic alteration in pmMRI, autopsy or histology. Table 1 displays the 19 lesions separated into three groups. Group 1 consisted of seven pmMRI lesions that correlated well to autopsy and histology. Group 2 included six cases that showed good correlation of pmMRI lesion to histology, but less or no correlation to macroscopic autopsy. There was a good correlation between pmMRI and histology in all group 1 and group 2 cases. Group 3 combined six cases that presented with lesions at pmMRI, but had no myocardial findings at autopsy or histology. All six group 3 cases were radiologically interpreted as peracute. These six peracute lesions presented with further cardiac autopsy findings that could explain an ischemic situation at the affected myocardial region by either severe coronary event/stenosis

Table 1 Findings of study cases grouped according grade of consensus

Case no.	Location	pmMRI	Autopsy	Histology	Cause of death	Comments
(a) Lesion group 1: consensus among pmMRI, autopsy and histology						
3	Anterior	Acute	Acute	Acute	Cardiac	Only small finding of 5×5 mm, no coronary finding
4	Anteroseptal	Acute and subacute	Acute and subacute	Acute and subacute	Cardiac	Severe LAD stenosis
8a	Anterior	Chronic	Chronic	Chronic	Cardiac	RCA + RCX occluded, LAD severe stenosis right after left main CA, bypass on RCA occluded, bypass on LAD open
8c	Lateral	Subacute	Subacute	Subacute		
14	Posteroseptal	Chronic	Chronic	Chronic	Cardiac	Severe 3 vessel disease with coronary bypass surgery
19	Lateral	Subacute	Subacute	Subacute	Cardiac	Severe RCX stenosis
20	Anterior	Acute	Acute	Acute	Cardiac	Severe LAD stenosis
(b) Lesion group 2: consensus between pmMRI and histology						
1a	Anteroseptal/ anterior	Subacute and chronic	Chronic	Subacute and chronic	Cardiac	Severe 3 vessel desease
1b	Lateral	Acute and subacute	Subacute	Acute and subacute		
7	Septal	Acute	-	Acute	Cardiac	Heart weight 613 g
12	Septal	Acute	-	Acute	Pulmonary/ cardiac	50% stenosis on LAD and severe pneumonia
13	Anteroseptal	Subacute and chronic	Chronic	Subacute and chronic	Cardiac	Severe stenosis at proximal LAD
16	Posteroseptal	Subacute	-	Subacute	Cardiac	Severe RCA stenosis
(c) Lesion group 3: no consensus between pmMRI and autopsy/histology						
2	Septal	Peracute	-	-	Cardiac	Severe stenosis at 2nd septal branch
6	Anteroseptal	Peracute	-	-	Cardiac	Severe stenosis at intermediate branch
8	b) Septal	Peracute	-	-	see case no. 8a	
10	Anterior/ant. papillary muscle	Peracute	-	-	Cardiac	Fresh ruptured soft plaque in LAD
17	Anteroseptal	Peracute	-	-	Cardiac	LV hypertrophy due to severe aortic valve stenosis
18	Anterior	Peracute	-	-	Cardiac	Severe LAD stenosis
(d) Study cases without myocardial findings in pmMRI, autopsy and histology						
5	-	-	-	-	Cardiac	Heart weight 690 g
9	-	-	-	-	Multiorgan failure	Pancreatic cancer
11	-	-	-	-	Cardiac	Heart weight 796 g
15	-	-	-	-	Pulmonary	Severe pneumonia

Grading of pmMRI findings and histological findings was assessed according to Jackowski et al. [19]. Autopsy diagnosis was based on the macroscopic appearance at autopsy only. The given cause of death represents the final integrative forensic case diagnosis including extra-cardiac autopsy findings. Case no. represents the initial chronological case numbering

(five cases) or severe myocardial hypertrophy (one case). Acute pulmonary edema and internal congestion indicated acute cardiac failure as the cause of death in all six group 3 cases.

The findings of chronic (Fig. 1), subacute (Fig. 2) and acute infarction (Fig. 3) demonstrated reproducible appearances in pmMRI. Chronic infarction was associated with a broad loss of signal in all applied sequences. In T2 weighting, subacute infarction displayed hyperintense alterations within the affected myocardium. Acute infarction showed a hypointense center with hyperintense margins because of edema in the outer zone in T2 weighting.

Discussion

In postmortem cardiac MR imaging, not having the possibility to assess late enhancements [24], excellent

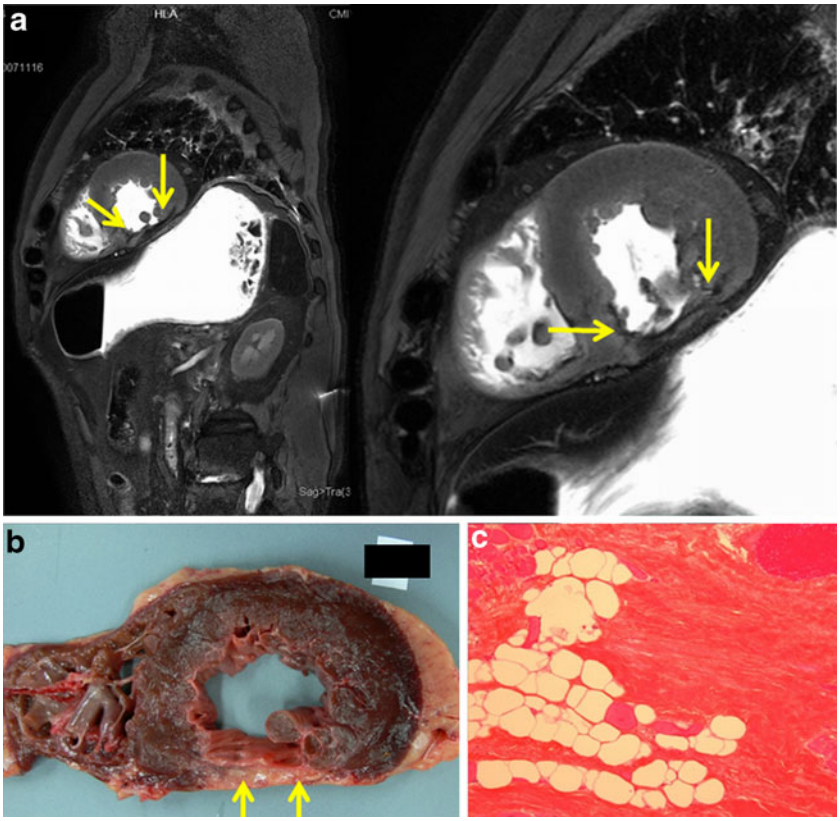

Fig. 1 Chronic myocardial infarction (case 14) in pmMRI. **a** T2-weighted short axis images (entire FoV and magnification) depict a severe shrinking of the inferior wall with broad decrease of signal (arrows). **b** Autopsy demonstrates definite collagenous transformation of the infarcted inferior myocardium with scar-caused shrinking. **c** Histology (H&E) shows cell-free collagen formation as the cause for the significant decrease of signal in MRI as well as fatty degeneration

image quality can nevertheless be obtained due to the absence of cardiac motion or breathing-related artifacts. The image contrast not based on perfusion differences visualized by contrast agent administration strongly represents morphological alterations within the myocardial tissue. Already in the early 1980s, unenhanced clinical MR imaging showed promising results for the identification of myocardial infarction in the living because of the increase of water (edema) within the infarcted muscle [25, 26]. Thereby, acute infarctions appear as hyperintense regions in in-vivo T2-weighted images and dark in native T1-weighted images [27].

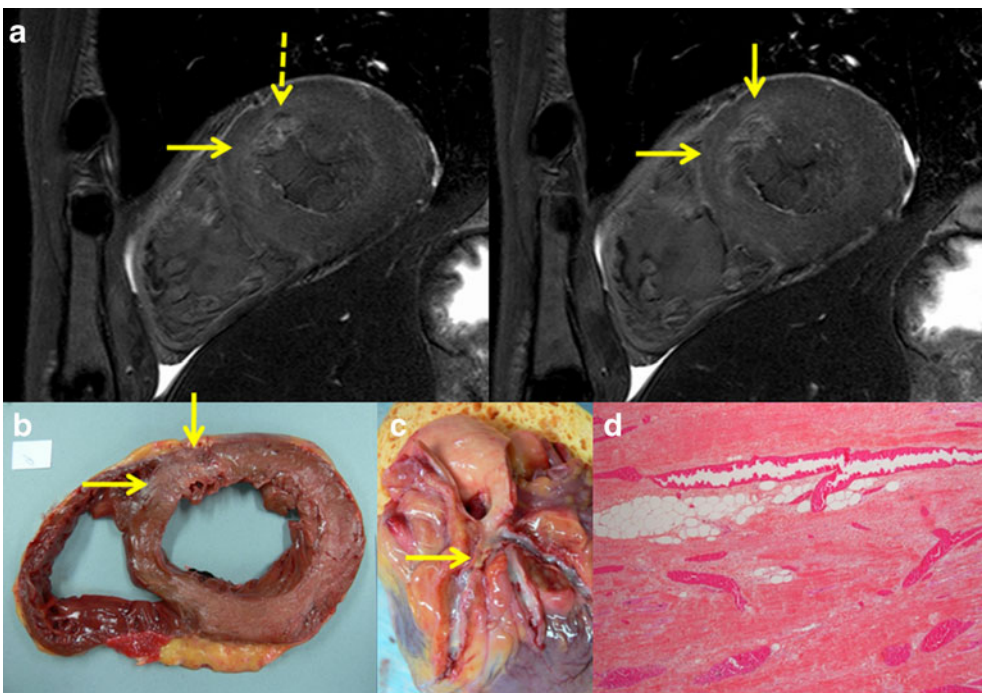

Fig. 2 Subacute myocardial infarction (case 13) in pmMRI. **a** T2-weighted short axis images at different levels show a broad hyperintensity (arrows) affecting the anteroseptal wall. Note a tiny spot of signal loss due to a small chronic infarction within the affected anterior wall (left image, dotted arrow). **b** Autopsy demonstrates discreet shrinking of the anteroseptal wall and minor connective tissue formation. **c** The coronary artery system presents with a severe stenosis at the LAD. **d** Histology (H&E) shows loose connective tissue formation and angiogenesis as the reason for the general signal increase in T2

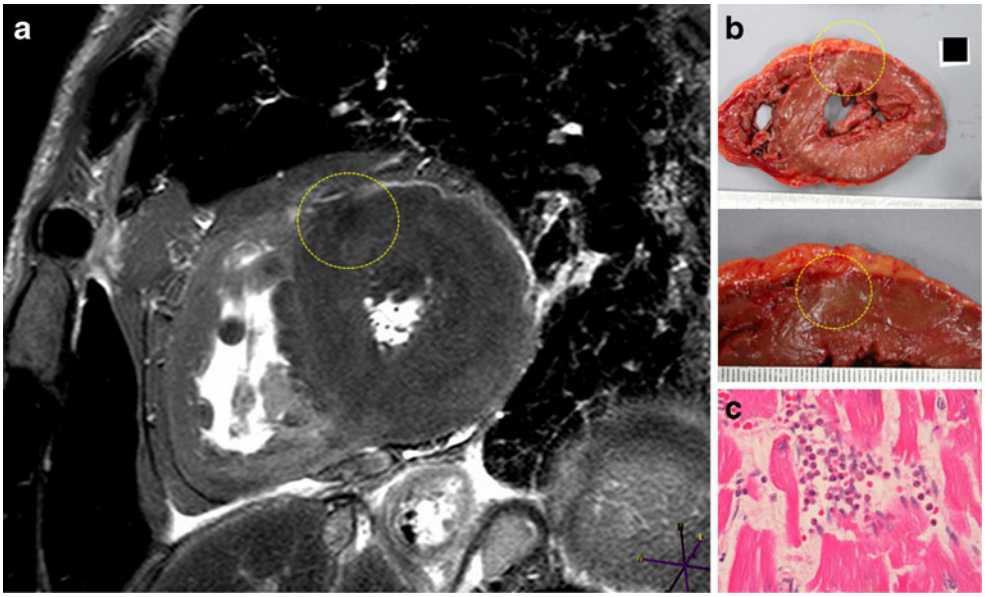

Fig. 3 Acute myocardial infarction (case 3) in pmMRI. **a** T2-weighted short axis image demonstrates a tiny hypointensity (circle) with indistinctive surrounding edema within the anterior wall. **b** Autopsy can reveal a small yellowish alteration at the same location (circle). **d** Histology (H&E) demonstrates beginning granulocyteous infiltration only in one magnification field as shown

Whereas clinical cardiac MR imaging in recent years has improved different contrast agents and implemented application schedules as well as contrast agent and cardiac motion-adapted sequences to better assess cardiac pathology [20, 28–31], postmortem cardiac MR imaging fully depends on imaging of unenhanced structural alterations at different stages of myocardial infarction. These stages showed a specific signal behavior in pmMRI that allows an age staging of the infarction. The present study confirmed the results from our recent study concerning the pmMRI findings in acute, subacute and chronic infarction [19].

Thirteen of the 19 lesions (group 1 and 2) correlated well between pmMRI and histology, whereas 6 of them (group 2) showed poor correlation to macroscopic autopsy. Macroscopic detection of myocardial infarction depends on the extent of discoloration and/or the consistency of the tissue. This makes overlooking tiny

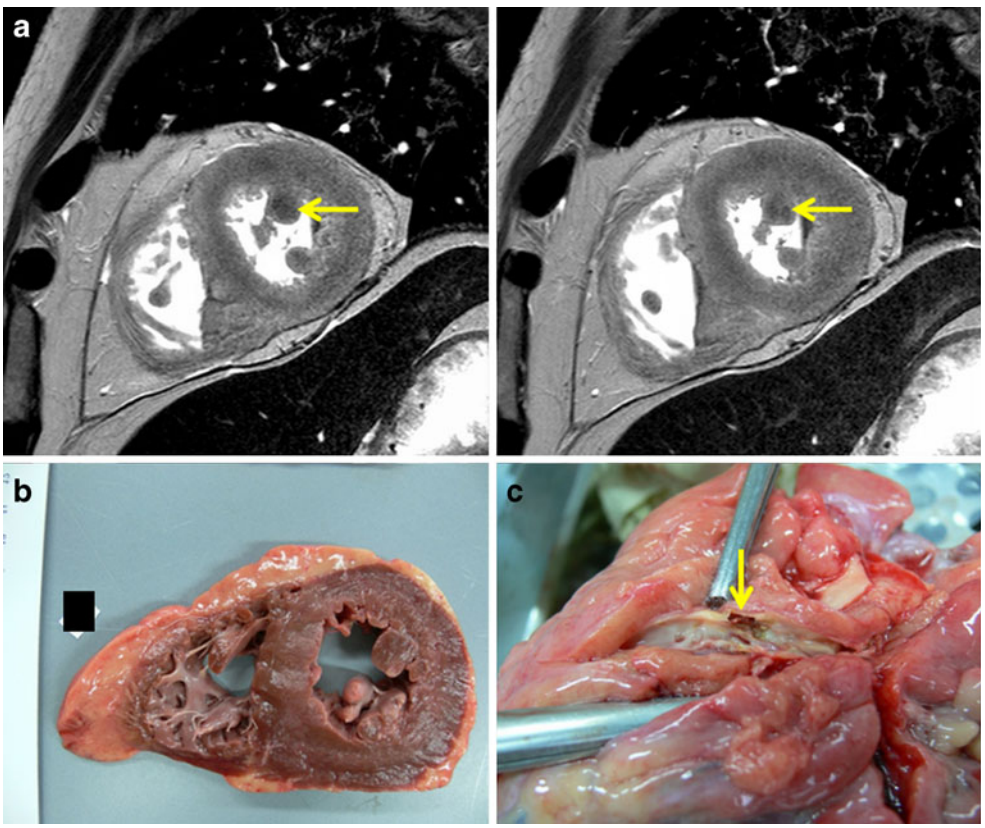

Fig. 4 Peracute myocardial infarction (case 10) in pmMRI. **a** T2-weighted short axis images at different levels of the anterior papillary muscle present with local hypointensities within the anterior papillary muscle and indistinctively also within the anterior wall. **b** Autopsy aspect of the specimen shows no visible alteration within the anterior wall and the papillary muscles. Histology also failed in demonstrating ischemic alterations (not shown). **c** Dissection of the coronary artery system reveals a fresh soft plaque rupture with intimal hemorrhage within the proximal LAD

lesions likely when no distinctive and distended alterations are present. Furthermore, an age staging by eye at the autopsy table is extremely uncertain, and histology is needed for validation. Therefore, the group 2 cases show exemplarily that pmMRI provides additional information in cases that are hard to diagnose macroscopically.

In addition to the recent work [19], the present study demonstrated that cases of sudden cardiac death displaying no myocardial morphological finding at autopsy and histology showed structural alterations in pmMRI (Fig. 4). These cases challenge pathologists since generations and all approaches to verify an ischemic situation before sudden cardiac death have not reached widespread application because of less satisfying performance [32–34]. In five of the six group 3 cases, myocardial pmMRI findings (hypointensities in T2 weighting) correlated well with coronary pathology at autopsy, whereas according to the literature [35] only half of the peracute cases present a coronary lesion at all. The sixth case revealed massive LV hypertrophy consistent with the possibility of a local relative ischemic incident. All peracute cases in our study group were forensically diagnosed as cardiac deaths in combination with indirect signs of cardiac failure such as internal congestion and acute pulmonary edema. Although statistically not supported because of the still rather small study population, the results of our pmMRI examinations let us assume that pmMRI is a suitable method to detect and display peracute myocardial lesions to be included into the case diagnosis.

In concordance with our previous work [19], acute myocardial infarction lesions were displayed as a zone with a dark ischemic center and a bright edematous margin (T2-weighted images). Since the development of post-infarct myocardial edema takes several minutes, it is not expected in sudden cardiac deaths due to an arrhythmic event resulting in a dark hypointense lesion representing the zone of ischemic myocardium without bright margin as observed in the six peracute cases. In conclusion, pmMRI seems to visualize ischemic myocardium well before the development of edema occurs. Within this short time period, the onset of a ventricular fibrillation may have caused cardiac failure and sudden death, preventing further vital morphological reactions in the myocardium.

The observation of hypointense areas in T2-weighted images in ischemic myocardial regions is not sufficiently explained yet. A slight decrease of signal also seen in PD-weighted sequences would suggest that the mean water content is lower than in the surrounding myocardium. A lower pH value in combination with local electrolyte changes may also contribute to a relevant T2 relaxation time shortening. However, the anatomical association of "dark myocardium" with a coronary lesion at the coronary artery perfusing the affected myocardium makes a causative correlation to an ischemic situation very likely. Compared to the in-vivo T2 appearance of acute infarctions, this is the most striking difference as clinical radiologists know acute infarctions as hyperintense in T2-weighted images. The edema as a vital myocardial reaction within the infarcted region is present from hours to days after the onset of ischemia. In the majority of cases when acute infarction is imaged postmortem, the patient died in very early stages. In these early time frames after ischemic onset (minutes to hours), the edema starts developing, and this stage is kept after death. Therefore, we see no edema in peracute cases (minutes) yet, but hypointense areas probably as a result of reduced microcirculation. In case the patient did not die immediately, we see the state of developing edema as a marginal ingrowing hyperintensity around the hypointensity in acute cases. Our hypothesis is that most of the in-vivo images are obtained at the stage of more or less fully developed edema and that postmortem images often show earlier stages with beginning edema or no edema.

Autopsy and histology, the "gold standard" to investigate cardiac deaths, are to be challenged by new approaches. However, without a coronary finding indicating a local circulation barrier, a peracute lesion causing an arrhythmic sudden cardiac death cannot be verified using traditional methods within the myocardium itself. The six cases in group 3 without consensus between pmMRI and autopsy and histology are not "false positive." It is the gold standard that obviously fails in these cases. Only the view of the coronary artery system helped to interpret the mismatch correctly. We think we have demonstrated that pmMRI may provide further valuable diagnostic information about the myocardium in peracute cardiac deaths; it is capable of visualizing where and how much distension the myocardium has suffered from absolute or relative ischemia.

Application of contrast agents may also be promising in postmortem imaging. Techniques of sufficient performance already exist and enable minimally invasive visualization of the coronary artery system [36–39]. In porcine ex vivo experiments, distribution defects of injected gadolinium could be simulated within the porcine myocardium, which possibly can be correlated with a disturbed antemortem perfusion as well [36]. A combination of pmMRI and minimally invasive angiography will enhance the diagnostic performance of pm imaging.

Limitations

The study was not conducted in a double-blinded way as radiological findings not detected at autopsy should undergo histological examination as well. Therefore, the forensic pathologist had to be informed about the pmMRI findings in advance to obtain tissue samples from both autopsy and pmMRI findings.

The study number is rather low, so a confirmation of our results in larger series is needed.

A further limitation may be the lack of a comparably sized control group with a sufficient number of non-cardiac deaths. The four study cases without a local myocardial finding may indicate a consensus for the

absence of myocardial findings, too. However, four cases do not allow for valid conclusions.

No measurements of relative signal intensities within the infarcted myocardium in comparison to skeletal or not affected myocardium are given. From the clinical point of view, this would be expected to lead to better understanding of the pm appearances of infarcted myocardium. However, the postmortem situation is more complex with respect to relative signal intensities. Both T1 and T2 relaxation are temperature dependent. The decrease of T2 relaxation time at decreasing temperature is less distinctive compared to the rather considerable decrease of T1 relaxation time at decreasing temperature. In contrast to the clinical situation where the patient's temperature is a predominantly stable value of about 37°C, we have to deal postmortem with a possible temperature ranging from about 4° to 40°C. Exceptions even lie beyond these values when, e.g., frozen bodies found in glaciers or burned bodies with still elevated core temperatures enter the MR machine. Therefore, any absolute quantified or relative signal intensity on postmortem MR data can only be interpreted with respect to the actual body temperature during data acquisition. As the body core temperature of the study cases was not consistent, it is not reasonable to compare absolute or relative signal intensities between different cases. To address this problem, a separate study is needed and already in preparation that will perform an absolute quantification of T1, T2 and proton density for every body tissue (grey matter, white matter, myocardium, liver tissue, etc.) at defined temperatures ranging from 4° to 40°C to compile a data collection that can be used on one hand for optimal sequence design and on the other hand for diagnostic purposes to characterize pathological alterations.

Conclusion

With pmMRI ischemic lesions can be detected, assessed and age staged. Indeed, in peracute lesions, as in sudden cardiac death when the lesion may not be visible with conventional autopsy techniques, it may provide additional information for the pathologist.

The present study demonstrates that pmMRI imaging provides an alternative and/or complementary postmortem examination technique to autopsy. Additionally, it should be considered for cases not undergoing a traditional autopsy. Depending on the country, up to more than 90% of the deceased do not undergo comprehensive postmortem examination. The negative consequences for medical education, quality assurance in medicine, public health and mortality statistics are substantial, as discussed by numerous authors [1–3, 5, 40]. Postmortem imaging has matured to a valuable postmortem examination method to acquire patho-anatomic details in a spatial resolution not achievable by routine autopsy. An increasing number of forensic institutes have started to install CT and MRI systems to use the imaging techniques for the purpose of quality improvement. However, it is the natural death that is significantly under-autopsied today, and it seems obvious to us that clinicians should become aware of the potential and the recent progress of pmCT and especially pmMRI as a non- or minimally invasive method for the investigation of the deceased patient. The aim is not to replace clinical autopsies, but to complement or offer an alternative when autopsy is not agreed to by the next of kin and to reestablish a reliable base of data of cause of death in our society.

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